Recurrent or New Symptomatic Cerebral Aneurysm after Previous Treatment

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Summary

With the establishment of endovascular coiling as a successful treatment for symptomatic cerebral aneurysms, attention is now being directed at the durability of this treatment. If this is to be accurately done it will be important to understand the causes of symptomatic aneurysm presentation after previous treatment. In order to assess this we undertook a retrospective review, covering the four year period from 2000 to 2004, of all patients re-presenting with a symptomatic saccular aneurysm after previous treatment. Seven patients were identified, six presenting with subarachnoid haemorrhage (SAH) and one with a third cranial nerve palsy. Three patients had incomplete clipping of their aneurysms and all presented within months of their initial treatment.

The other four patients presented between five and 20 years after primary treatment and all were felt to have new cerebral aneurysms. Two of these patients had aneurysms develop at the same location as their previously treated lesions, however these were still felt to be new aneurysms rather than re-growth or recurrence because of their morphology. Based on our findings it would appear that development of a new cerebral aneurysm after clipping is more of a risk than aneurysm recurrence from treatment failure. This will need to be considered when evaluating re-presentation after treatment by either coiling or clipping and more importantly, perhaps we should be directing more attention to preventing disease progression rather than treatment failure.

Introduction

The surgical and endovascular treatment of cerebral saccular aneurysms appears to offer a stable long-term outcome. Reported incidence of re-presentation of patients with subarachnoid haemorrhage after successful treatment vary from 0 to 2.5% over follow-up periods that cover epochs of five to 20 years [1,2]. There are however reports of aneurysm re-growth after incomplete or complete treatment and reports of de novo aneurysm formation [3,4]. Most reports relate to the follow-up of surgical series primarily because micro-surgical clipping has been a standard treatment for over 40 years. Attention is however now being focused on the durability of endovascular coiling as a therapy. Comparisons of available data are however not easily done as most series fail to differentiate between the types of aneurysms treated, what constitutes a complete treatment and what is seen as a failure of therapy, i.e. aneurysm re-growth or re-bleeding. There has also been a focus on the incidence of failure rather than the reasons for treatment failure. We undertook a retrospective review of patients presenting within the last eight years with either SAH or

cranial nerve palsy who had been previously treated for a symptomatic saccular aneurysm. Our aim was to define the causes of re-presentation, separating failure of therapy (symptomatic re-growth or rupture) from progression or evolution of the disease (development of a new aneurysm).

Methods

A review of our neurovascular database was undertaken searching for patients presenting with either SAH or a new cranial nerve palsy who had previously had treatment of a saccular aneurysm. The period reviewed was from January 2000 to December 2004. Patients with giant aneurysms, dissections or infective aneurysms were excluded. Primary presentation could have been at any prior time, either days or years before their new presentation. Once identified, the patients clinical notes, procedure notes and radiological images were reviewed. In each case we endeavoured to define the reason for their treatment failure. During the four year period of this review endovascular therapy has been available however prior to 2000 only surgical clipping was available as a treatment for cerebral aneurysms. After surgical clipping it has not been our routine to repeat angiography unless the surgeon thought the aneurysm had not been completely clipped. Patients who were treated with coiling were routinely followed up with angiography at three months and one year. Accurate figures for the number of patients treated since the first presentation of a patient were not available and consequently we could not derive a figure for the annual risk of treatment failure.

Results

Seven patients were identified who re-presented after they had received treatment for a saccular aneurysm. All of these patients had been surgically treated with aneurysm clipping. Three patients were classified as having incomplete aneurysm clipping and all re-presented within months of their initial surgery. New aneurysms were felt to be the cause of re-presentation in a further three patients, two occurring at a similar location to the primary aneurysm and one in a remote location. These patients presented years after their initial treatment. One patient had growth of a mirror aneurysm from a small bleb to a symptomatic aneurysm. Brief case details for each patient are as follows.

Case 1

A 36-year-old male patient presented in 1997 ten days after suffering a SAH. On CT scan he had a clear interhemispheric haemorrhage and angiography confirmed there was a 3 mm acom aneurysm filling from the right carotid injection. The aneurysm was surgically approached and the operation note recorded that there was some difficulty defining the aneurysm due to it being partially thrombosed. It was however thought to be clipped. After recovery the patient was discharged home but presented again one month later with another SAH. Repeat angiography demonstrated severe vasospasm and showed that the aneurysm was not completely clipped. Further surgery was performed and the aneurysm completely clipped. The patient recovered but was left with a left sided hemiparesis and required a shunt for hydrocephalus.

Case 2

A 21-year-old male presented in 2001 with a WFNS grade II SAH. The origin of the bleed was thought to be from a ruptured anterior communicating (acom) artery aneurysm which was seen on MRA and angiography. The aneurysm was clipped with some intra-operative difficulty. Despite applying two clips to occlude this aneurysm there was felt to be a residual thin bleb involving the right A2 vessel. A post-operative angiogram however was thought to be satisfactory and the patient was discharged. He presented again four months later with SAH. All the images were reviewed and the A2 aneurysm was felt to be significant. Re-exploration was performed and the A2 aneurysm was defined and clipped. The patient made a full recovery.

Case 3

A 38-year-old female presented in 2004 with a WFNS grade 3 SAH. An angiogram demonstrated a 3 mm acom aneurysm with vasospasm involving the left internal carotid artery. The aneurysm was felt to be unsuitable for coiling primarily because severe vasospasm would make access difficult. After clinical improvement 13 days later surgery was performed. This was complicated by intra-operative aneurysm rupture and difficulty controlling bleeding despite

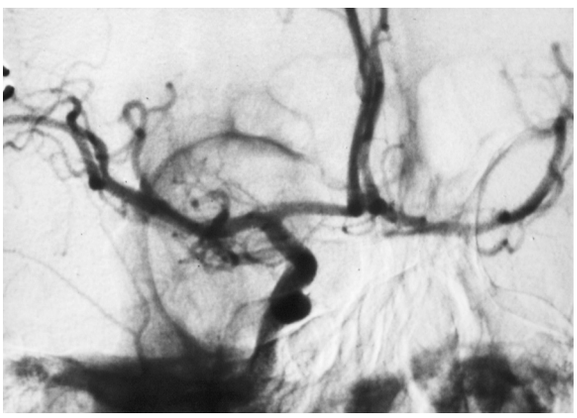

Figure 1 Pre-operative angiogram of Patient 1 showing a 3 mm acom aneurysm.

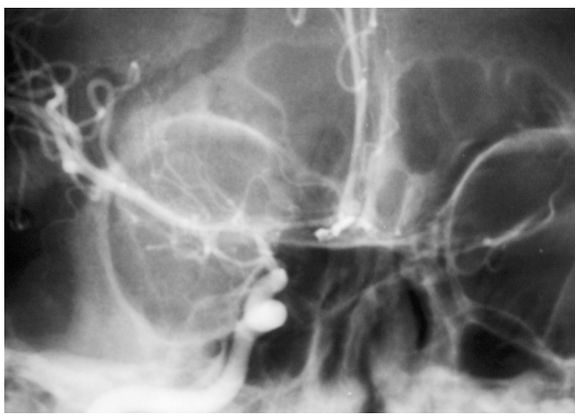

Figure 2 Postoperative angiogram of Patient 1 with the clip not occluding the neck.

clip placement across the neck. Eighteen days after surgery the patient suffered another SAH. Angiography demonstrated that the neck of the aneurysm had not been clipped and most of the fundus was still open. A decision was made to coil the aneurysm rather than attempt clip repositioning. This was accomplished without complication. The patient recovered and was discharged home.

Case 4

A 42-year-old female presented in 2002 with a WFNS grade I SAH. She had previously had surgical clipping of a ruptured left middle cerebral artery (mca) aneurysm in 1982. An angiogram demonstrated a large M1 middle cerebral artery aneurysm adjacent to the original clip. Because the clip was un-displaced and still against the mca vessel wall this was felt to be a new aneurysm and not re-growth of the previous aneurysm. The new aneurysm was coiled and the patient recovered well.

Case 5

A 28-year-old female presented in 2003 with a painful right ptosis. She had received surgical clipping in 1983 for a right pcom aneurym after suffering SAH. An angiogram demonstrated a large right pcom aneurysm just proximal to the previous clip. Again this was felt to represent a new aneurysm rather than recurrence due to the clip apposition to the internal carotid. The aneurysm was coiled and occluded with complete recovery of the III nerve palsy.

Case 6

A 42-year-old female presented in 2004 with grade I SAH. In 1999 she had also had SAH

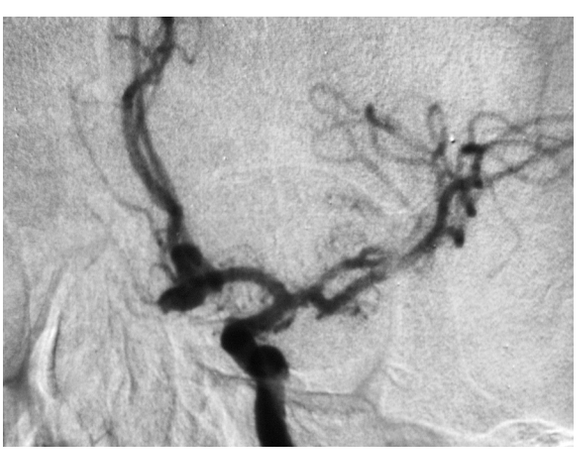

Figure 3 Patient 2 pre-operative angiogram showing a complex acom aneurysm.

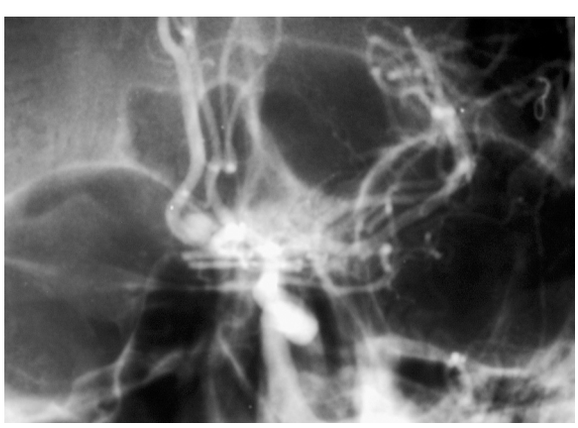

Figure 4 Patient 2 postoperative angiogram showing occlusion of the inferior component of the aneurysm but filling of the superior part.

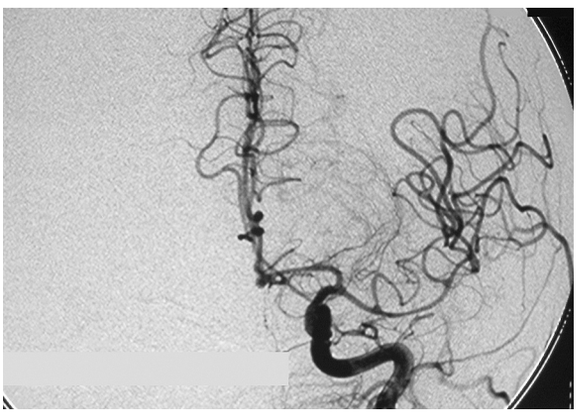

Figure 5 Patient 3 pre-operative angiogram showing severe vasospasm and a small acom aneurysm.

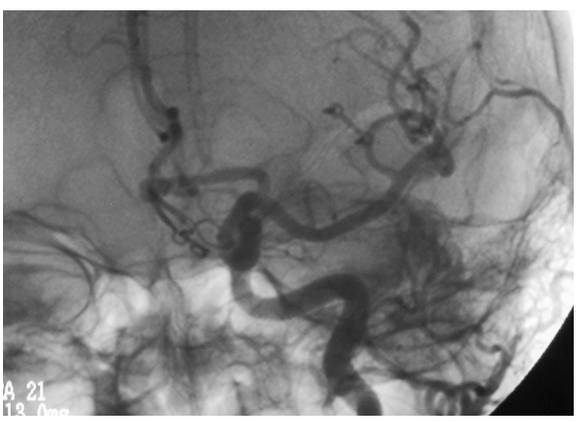

Figure 6 Postoperative angiogram of patient 3 with the clips not occluding the aneurysm neck.

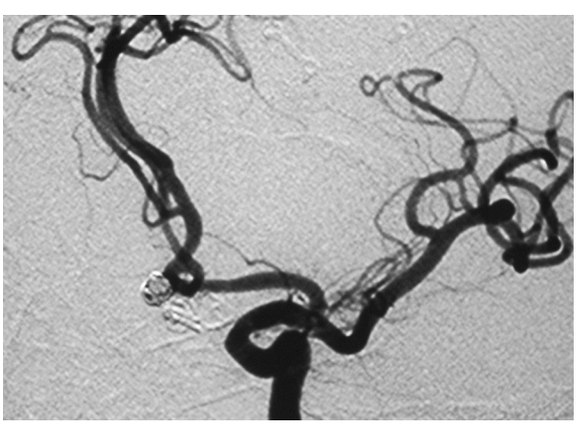

Figure 7 Patient 3 with complete occlusion of the aneurysm after coiling.

and at that time had surgical clipping of a ruptured left pcom aneurysm as well as a left anterior choroidal aneurysm and a pericallosal aneurysm. Her new angiogram demonstrated complete clipping of these aneurysms but the development of a new 3 mm basilar tip aneurysm and a small distal pericallosal aneurysm. These were not present on the initial angiogram. Her basilar aneurysm was coiled and she made a complete recovery.

Case 7

A 43-year-old female presented in 2003 with a painful right III nerve palsy. She had previously had clipping of a ruptured left pcom aneurysm in 1998. On her initial angiogram in 1998 she was noted to have a small carotid bleb in the region of the right pcom artery. On her new angiogram this bleb had enlarged to a 5 mm aneurysm which was coiled resulting in recovery of her ophthalmoplegia.

Discussion

Patients in our series clearly fall into two groups, those presenting with SAH within months of treatment and those presenting with symptomatic aneurysms years later. The early group had incompletely clipped aneurysms often related to difficult surgery or intra-operative rupture. Very little is reported in the literature regarding these early failures although in the ISAT trial ten patients in the surgical group had a post clipping haemorrhage within the first year after treatment [5]. Six of these patients bled within 30 days and of these three had incompletely clipped aneurysms like our patients. The remaining three patients were assessed as having completely clipped aneurysms although there are no details regarding the difficulty of surgery or post surgery angiography. In all of our cases we were able to complete the aneurysm treatment after re-haemorrhage without complication. This may not always be possible however as some aneurysms cannot be completely clipped. It is our impression however that partial clipping associated with intra-operative rupture and the development of a false aneurysm poses the greatest risk to the patient.

Patients presenting with a late haemorrhage in our experience are more likely to bleed from a new aneurysm rather than the treated aneurysm. Other authors have however been reluctant to think of an aneurysm developing in the region of a previously treated aneurysm as new, preferring to call these recurrences [6,7,8].

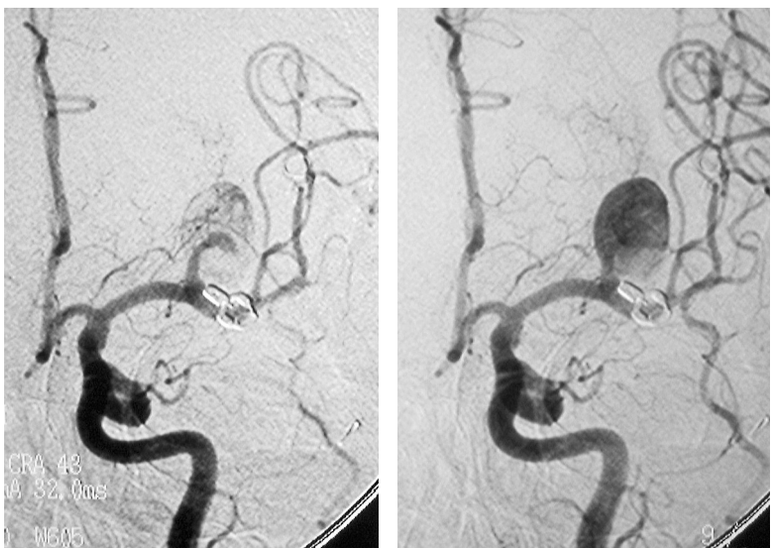

Figure 8 Angiogram of patient 4 showing the previously placed clip adjacent to the neck of a large new middle cerebral artery aneurysm.

This is despite the description of an aneurysm developing in the location of a previously excised aneurysm [3]. There are also descriptions of aneurysms developing at the same site despite post surgery angiography showing complete aneurysm occlusion [4,6]. Reasons for calling these recurrences rather than new or de novo aneurysms are that they represent residual neck enlargement, a slipped clip or growth of aneurysmal rests [9,10]. Even these authors admit however that slipping of a surgical clip placed over the neck of a small aneurysm is very unlikely. Of course growth of a residual aneurysm neck does represent a failure of treatment and is re-

Table 1 **Summary of patient details.**

Patient	Initial Aneurysm	Initial Treatment	Delay to re-presentation	Aneurysm at re-presentation	Reason for re-presentation	Second treatment
1	acom	Clipping	1 mth	acom	Re-bleeding after partial clipping	Clipping
2	acom	Clipping	4 mth	acom	Re-bleeding after partial clipping	Clipping
3	acom	Clipping	13 days	acom	Re-bleeding after partial clipping	Coiling
4	L mca	Cipping	10 yr	L mca	Haemorrhage from new aneurysm	Coiling
5	R pcom	Clipping	20 yr	R pcom	Haemorrhage from new aneurysm	Coiling
6	L pcom L Ach Pericallosal	Clipping	5 yr	Basilar tip	Haemorrhage from new aneurysm	Coiling
7	L pcom	Clipping	5 yr	R pcom	III Nerve palsy from new aneurysm	Coiling

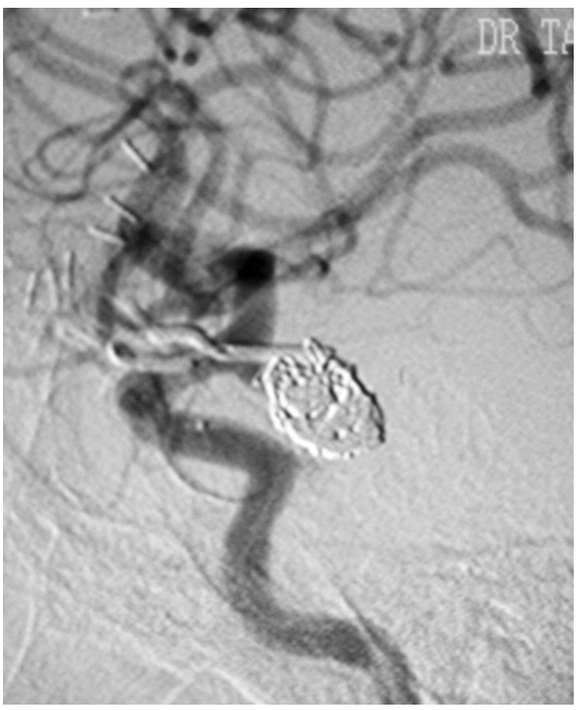

Figure 9 Patient 5 angiogram with a new aneurysm, which was coiled, immediately adjacent to the clip placed 20 years previously.

ported to occur in 2.9% of cases where the aneurysm was not completely clipped. Based on our observations however we feel that neck enlargement should lead to some degree of clip displacement from the parent vessel. Where this does not occur the aneurysm should be considered new. In our series three patients out of four developed new aneurysms after treatment according to this definition: one in a remote location and two at the site of previous treatment. It seems likely that the region of vessel where an aneurysm develops is vulnerable and that this area of vulnerability extends over a larger area than just the aneurysm neck. Consequently even if an aneurysm is completely treated the distal and proximal parent vessel wall must still be at risk of future aneurysmal development. Vessel wall vulnerability may also extend to the mirror vessel segment on the contra lateral side in certain patients, and this would account for the observed aneurysm growth over a five-year period in patient seven. New aneurysm development at a remote location to previously treated aneurysms is more likely in young female patients with multiple aneurysms [6,11]. Patient six in our series supports

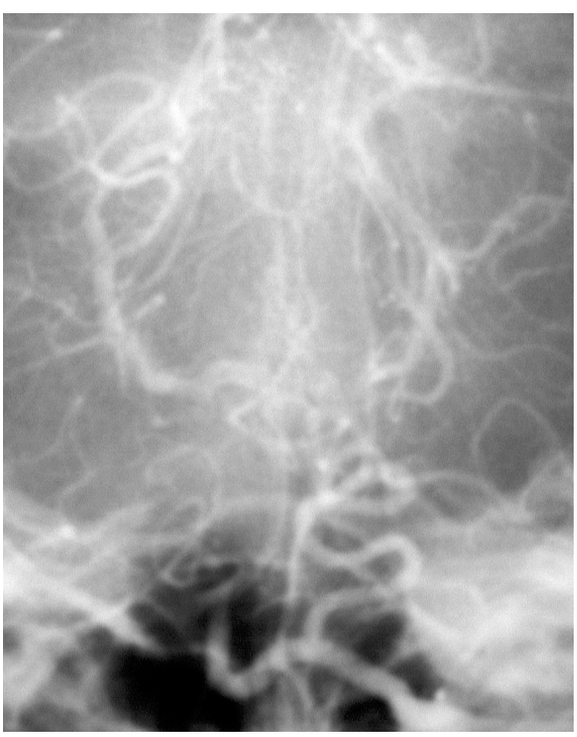

Figure 10 A poor quality vertebrobasilar angiogram of patient 6 at her first admission showing no aneurysm.

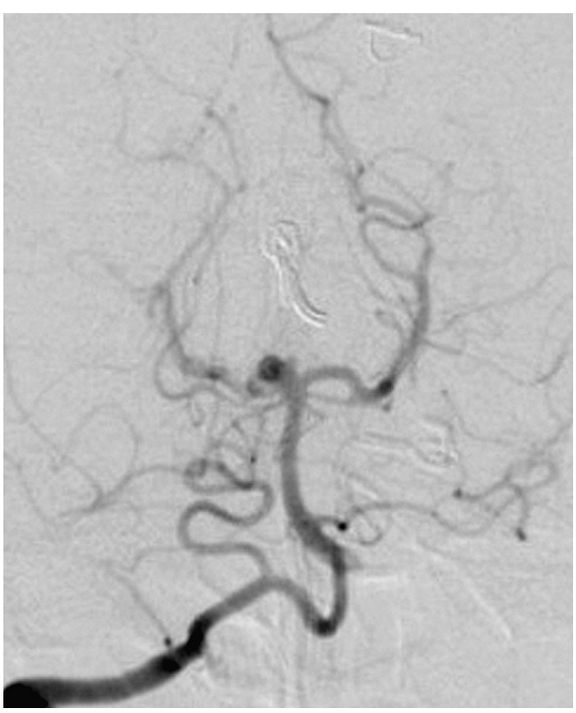

Figure 11 Patient 6 vertebrobasilar angiogram repeated five years later after she presented with an interpeduncular subarachnoid haemorrhage showing a small basilar tip aneurysm.

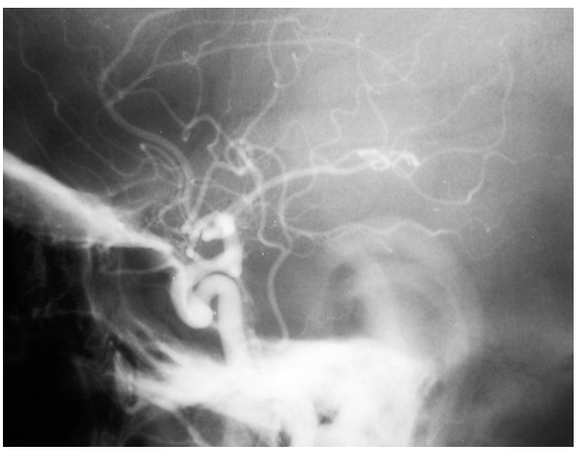

Figure 12 Patient 7 left carotid angiogram in 1998 showing a small carotid bleb near the pcom origin.

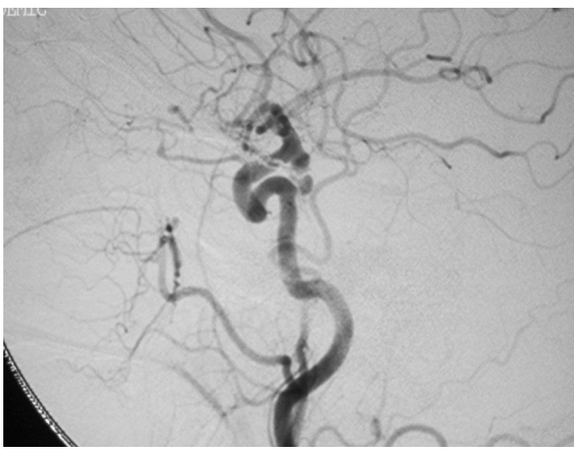

Figure 13 Patient 7 left carotid angiogram in 2003 showing an irregular typical pcom aneurysm. The visible clip is on the right side.

these findings and it would appear that this group of patients have a vessel wall vulnerability that extends to multiple locations and allows for early clinical expression.

In our experience we have not had any coiled saccular aneurysm patients present with a post treatment haemorrhage. Patients are perhaps protected from early haemorrhage as incomplete treatments are detected immediately and either coiled further or surgically treated. After only four years of follow-up it is also too early to expect late haemorrhage from this coiled group.

Conclusions

Failure of aneurysm treatment can occur early and is most likely due to an incompletely treated aneurysm. Late failure however is more likely to be due to the development of a new aneurysm. The new aneurysm may be at a remote location but development at the site of prior treatment does not preclude this from being a new lesion. Before comparisons are made between the durability of surgical or endovascular treatments it will be necessary to clearly define not only the nature of the aneurysms being treated but also the reasons for treatment failure.

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EDITORIAL COMMENT

The issue of de novo aneurysm is very important both after clipping and after coiling. This may reflect insufficient screening at the time of the initial angiogram and therefore may account for the variability of multiple aneurysms reported in most clinical series. In Bicêtre, a series dealing with 462 consecutive patients between 1993 and 1999, 26.4% actually presented more than one lesion: 122 patients for a total of 305 aneurysms. The discrepancy in the literature ranges from 7.7 to 44.9% depending on the series.

Following up our series of patients treated with coiling, the risk of developing de novo aneurysms was 0.76% per patient year. This certainly points to the segmental vulnerability of the cervicocranial and cerebral vasculature and also to the need for accuracy in the assessment of healed targets versus the recurrence within vulnerable areas.

In all the cases in which de novo aneurysm was present, the appearance of the lesion or its symptomatic character was noticed six years after initial treatment.

P. Lasjaunias, M.D.

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